Motion Sickness: Diagnostic Criteria
Consensus document of the Classification Committee of the Bárány Society

*Yoon-Hee Cha¹, John Golding², Behrang Keshavarz³, Joseph Furman⁴, Ji-Soo Kim⁵, Jose A. Lopez-Escamez⁶, Måns Magnusson⁷, Bill J. Yates⁸
Advisers: Jeffrey Staab⁹ and Alexandre Bisdorff¹⁰

¹Department of Neurology, University of Minnesota, Minneapolis, MN., USA
²Department of Psychology, University of Westminster, London, England
³Toronto Rehabilitation Institute – University Health Network, Toronto, ON, Canada; Department of Psychology, Ryerson University, Toronto, ON, Canada
⁴Department of Neurology, University of Pittsburgh, Pittsburgh, PA., USA
⁵Department of Neurology Seoul National University, Seoul, Republic of Korea
⁶Otology and Neurotology Group CTS495, Department of Genomic Medicine, Centre for Genomics and Oncology Research – Pfizer/Universidad de Granada/Junta de Andalucía (GENyO), PTS, Granada, Spain
⁷Department of Otolaryngology, Lund University, Lund, Sweden
⁸Department of Otolaryngology, University of Pittsburgh, Pittsburgh, PA., USA
⁹Department of Psychiatry and Psychology, Mayo Clinic, Rochester, MN., USA
¹⁰Department of Neurology, Centre Hospitalier Émile Mayrisch, L-4005 Esch-sur-Alzette, Luxembourg

Abstract
We present diagnostic criteria for motion sickness to be included in the International Classification of Vestibular Disorders. Motion sickness is a normal physiologic response that can be elicited in almost all individuals, but susceptibility and severity can be high enough to be considered a disorder in some cases. Motion sickness may be induced by motion of the head or by motion of the visual field. Sickness susceptibility to each motion stimulus type may vary within individuals.

An episode of motion sickness may be diagnosed when all of the following criteria are met: A. One or more of the following symptoms triggered by passive or active head motion: nausea, stomach awareness, sweating, headache, or drowsiness, B. Symptom or symptoms develop during the motion exposure, C. Symptom(s) onset is gradual, D. Symptom(s) eventually stop after cessation of motion, E. Not better accounted for by another disease or disorder.

A motion sickness disorder (MSD) may be diagnosed when all of the following criteria are met: A. Recurrent episodes of motion sickness triggered by the same class of motion stimuli (Definite MSD=five episodes, Probable MSD=two to four episodes), B. Symptoms are reliably triggered by the same class of motion stimuli, C. Symptom severity does not significantly decrease despite repeated exposure to the same class of motion stimuli, D. Symptoms lead to one or more of the following behavioral responses: a. Activity modification to abort sickness symptoms, b. Avoidance of motion stimuli that trigger sickness, c. Negative anticipatory emotions before exposure to motion stimuli, E. Not better accounted for by another disease or disorder.
Disorder (VIMSD) is made when the triggering stimulus is visual motion. An individual may be diagnosed with MSD, VIMSD, or both.

1. Introduction
The Classification Committee of the Bárány Society was charged with developing diagnostic criteria for motion sickness. These criteria were developed by an international group of vestibular specialists, scientists, and therapists for the Bárány Society in order to promote a common reference frame across disciplines involved in the care of the general public and in individuals with increased susceptibility to motion sickness. Though a common phenomenon, sickness induced by head or visual motion can adversely affect otherwise healthy individuals and present serious safety issues in situations that require high levels of attention. The establishment of these criteria recognizes that motion sickness, while being a normal physiologic response, can have profound negative effects when severe.

1.1 History
Descriptions of motion sickness date back to 400BCE and have trended with evolution in modes of transportation [1]. Theories to explain this bothersome phenomenon evolved over the centuries, including dysequilibrium of the four humors (yellow bile, black bile, phlegm, blood), abnormal flow of qi (life force), coordination of sensory inputs through the eyes and ears, and the effects of motion on inner ear function [1]-[3].

Interest in the mechanisms, treatment, and prevention of motion sickness had been historically driven by the military as they faced practical aspects of long-distance transportation of personnel in caravans, ships, and planes. The development of the space program in the 1960’s further spurred interest in motion sickness research, creating a foundation of clinical and physiologic assessment tools on which modern motion sickness research developed [4].

More recently, motion sickness that is mainly caused by stimulation of the visual system in the absence of physical movement has received increased recognition [5][6]. Scientific reports of visually induced motion sickness (VIMS) were first documented in the military domain when flight simulators were found to cause motion sickness-like symptoms [7]-[9]. Due to technological advancements over the past decades, visual displays and applications have become pervasive (smart phones, tablets, simulators, and virtual reality glasses), making VIMS a common phenomenon that affects a broader population.

1.2 Theories
Different theories exist for the generation, consequence, and modulation of motion sickness and VIMS (see Golding et al., 2016 and Keshavarz 2014, for discussions [5][10]) [4][11]-[13]. The sensory conflict and mismatch theories of motion sickness are arguably the most common explanations; they pertain to the conflict between expected versus actual visual, vestibular, and somatosensory inputs [4][11]. This conflict can be due to dissonance among sensory systems [14], mismatch between canal and otolith inputs (reviewed in [15]), or a discrepancy between perceived and expected verticality [16]. Motion sickness may be a by-product of the activation of vestibulo-autonomic pathways by head motion or visual stimuli to which the human nervous system is unable to or has not had sufficient time to adapt [5][17][18]. These theories predict that motion sickness would diminish with repeated exposure as the internal model of expected sensory input updates with new experiences.
In contrast, the toxin theory of motion sickness postulates that motion induced nausea and emesis may be evolutionarily protective responses to visual-vestibular mismatch that resembles the ingestion of poisonous food [19]. Consequently, the organism responds with retching and vomiting to expel the poison. A shared susceptibility to triggering the emetic pathway by different mechanisms is supported by the observation that individuals with high motion sickness susceptibility are more likely to suffer from nausea and vomiting from other causes, such as chemotherapy and actual toxins [20]. Motion sickness may also be an unfortunate consequence of the proximity of anatomical pathways that mediate vestibular signals with nausea and vomiting, without conferring any functional benefit to the individual. [10][21].

2. Methods
Members of the Classification Committee of the Bárány Society (CCBS) who met in Berlin, Germany in March 2017 proposed the creation of a subcommittee to develop criteria for motion sickness for the International Classification of Vestibular Disorders (ICVD). They selected a Chairperson (YHC) to choose subcommittee members. Subcommittee members were chosen to represent the fields of neurology, otolaryngology, psychology, rehabilitation, and vestibular physiology with committee members representing at least three continents. Communication among subcommittee members and between the Chairperson and individual committee members occurred through email and personal communication at meetings. The CCBS met again before the 30th Bárány Society meeting in Uppsala, Sweden in June 2018 and in Berlin, Germany in November 2019 to discuss progress. Diagnostic criteria were developed through discussions among subcommittee members. Draft criteria were presented to the CCBS in November 2019 and then modified based on comments. A revised draft was made available for comment by the Bárány Society membership in January 2020. Further comments and concerns were addressed before submission for publication.

3. Diagnostic Criteria for Motion Sickness
Motion sickness is a poly-symptomatic syndrome that is influenced by factors such as individual susceptibility (age and sex), stimulus type (self or visual motion), and specific circumstances (biological rhythms). The development of these criteria acknowledges the large body of observational, interventional, epidemiologic, and physiological studies that have studied the contribution of these factors to motion sickness severity and susceptibility [22]-[25].

3.1 Criteria for an Episode of Motion Sickness
An episode in which Criteria A through E are met:
A. One or more of the following symptoms triggered by passive or active head motion:
  1. Nausea
  2. Stomach awareness
  3. Sweating
  4. Headache
  5. Drowsiness
B. Symptom or symptoms develop during the motion exposure
C. Symptom(s) onset is gradual
D. Symptom(s) eventually stop after cessation of motion
E. Not better accounted for by another disease or disorder
NOTES:
1. A variety of motion stimuli can trigger motion sickness but susceptibility to one type of stimulus does not necessarily correlate with susceptibility to others. Trigger subtypes may include but are not limited to the following:
   a. Water transportation, e.g., boats, rafts, snorkeling, scuba-diving, and docks
   b. Air transportation, e.g., airplanes, helicopters, hang-gliding, parasailing, and parabolic flight
   c. Land transportation, e.g., cars, trains, buses, other land-based vehicles, and animal-based transportation
   d. Other moving platform, e.g. amusement park rides, waterbeds, and treadmills.
   e. Low frequency sway of the tops of very tall buildings
   f. Vestibular stimulation in laboratory settings, e.g., off-vertical axis rotation (OVAR), horizontal or vertical low frequency linear oscillation, and human centrifuges
   g. Space travel, e.g., orbiting, launching, re-entry.

2. Nausea may be mild or severe. Though the probability of vomiting is generally related to the severity of nausea, vomiting may also occur at lower levels of nausea in some individuals. These cases are uncommon, however.

3. Stomach awareness is an early manifestation of increased interoceptive awareness in motion sickness. Other less common manifestations include awareness of breathing and heartbeat; other visceral or somatic perceptions may be present, however.

4. Sweating in motion sickness is generally a ‘cold’ sweat that can accompany other autonomic symptoms and signs such as pallor, increased salivation, or decreased gastric motility.

5. Increased motion sickness susceptibility occurs in individuals with migraine or vestibular migraine [26] (See section 4.5.1) but headaches triggered by motion are not necessarily migraine headaches.

6. Arousal changes may result in drowsiness, yawning, sleepiness, or lethargy. These symptoms have historically been referred to as the ‘sopite’ syndrome [27].

7. Though some symptoms of motion sickness may persist after termination of the stimulus, the onset of sickness symptoms must occur during the motion stimulus and not exclusively after the stimulus has ended. This distinguishes motion sickness from mal de débarquement syndrome (MdDS), which only begins once the motion has ended and lasts for at least 48-hours [28].

8. The onset of sickness symptoms occurs gradually and generally after some delay relative to the onset of the sickness inducing motion. Symptoms that start immediately with head movement or are maximal at onset should raise suspicion for a structural vestibular disorder. Though motion sickness gradually increases with longer duration of exposure, severity starts to plateau with time. If vomiting is to occur, it generally happens within 60-minutes of the onset of other symptoms, such as nausea [17][29]. Continued exposure may also lead to habituation and eventual reduction of sickness.

9. Recovery from motion sickness is usually rapid after the cessation of motion. Severity should be less than 50% of the peak intensity within 1 hour of the cessation of motion. Some degree of residual symptoms may persist for hours after the cessation of motion, but generally abate within the day. An
exception is made for headache, which can last until specifically treated. In rare cases, an episode of motion sickness can lead to persistent symptoms despite the cessation of motion. These cases should prompt a search for secondary causes of symptoms that can overlap with motion sickness.

10. Motion sickness may coexist and can be impacted by the presence of vestibular disorders such as vestibular migraine, vestibular neuritis, or persistent postural perceptual dizziness. In those situations, both a diagnosis of motion sickness and the contributing disorder should be made. Motion sickness should not be equated with motion-induced vertigo. Vertigo is specifically defined as a “sensation of self-motion when no self-motion is occurring or the sensation of distorted self-motion during an otherwise normal head movement,” [30]. Though clinical entities that cause vertigo can also cause symptoms similar to motion sickness, motion sickness inducing stimuli do not cause an altered sensation of self-motion. When an episode of vertigo leads to overlapping symptoms with motion sickness, these symptoms are considered sequelae of the vertigo spell and not a separate motion sickness syndrome.

3.1.2 Criteria for an Episode of Visually Induced Motion Sickness (VIMS)
An episode in which Criteria A through E are met:
A. One or more of the following symptoms triggered by visual motion1:
   1. Nausea²
   2. Stomach awareness³
   3. Sweating⁴
   4. Headache⁵
   5. Drowsiness⁶
   6. Eye strain/blurred vision⁷
B. Symptom or symptoms develop during the visual motion exposure⁸
C. Symptom(s) onset is gradual⁹
D. Symptom(s) eventually stop after cessation of visual motion¹⁰
E. Not better accounted for by another disease or disorder¹¹

NOTES:
1. Visual stimulation may be in either laboratory or real-life settings, e.g. virtual reality, simulators, movies, tablets, smartphones, computers, video games, and optokinetic drums. When visually triggered motion sickness also includes head motion, e.g., motion-based flight simulators, driving simulators, ship simulators, the contributions of both stimuli should be noted.

2. Nausea may be mild or severe. Though the probability of vomiting is generally related to the severity of nausea, vomiting may also occur at lower levels of nausea in some individuals. These cases are uncommon, however.

3. Stomach awareness is an early manifestation of increased interoceptive attention in VIMS. Other less common manifestations include awareness of breathing and heartbeat; other visceral or somatic perceptions may be present, however.

4. Sweating in VIMS is generally a ‘cold’ sweat that can accompany other autonomic symptoms and signs such as pallor, increased salivation, or decreased gastric motility.
5. Headache is more common and may occur more frequently without nausea in VIMS than in motion sickness due to movement of the head [5][6].

6. Arousal changes may result in drowsiness, yawning, sleepiness, or lethargy. These symptoms have historically been referred to as the ‘sopite’ syndrome [27].

7. Eyestrain and/or visual blurring are particularly common symptoms of VIMS [5][6][24].

8. Though some symptoms of VIMS may persist after termination of the stimulus, the onset of sickness symptoms must occur during the visual motion stimulus and not exclusively after the stimulus has ended.

9. The onset of sickness symptoms occurs gradually and generally after some delay relative to the onset of the sickness inducing motion. Symptoms that start immediately with visual motion should raise suspicion for a primary ocular disorder. Though VIMS gradually increases with longer duration of exposure, severity starts to plateau with time. If vomiting is to occur, it generally happens within 60-minutes of the onset of other symptoms, such as nausea [17][29]. Continued exposure may also lead to habituation and eventual reduction of VIMS.

10. Recovery from VIMS is usually rapid after the cessation of visual motion. Severity should be less than 50% of the peak intensity within 1 hour of the cessation of visual motion. Some degree of residual symptoms may persist for hours after the cessation of visual motion, but generally abate within the day. An exception is made for headache, which can last until specifically treated. In rare cases, an episode of VIMS can lead to persistent symptoms despite the cessation of visual motion. These cases should prompt a search for secondary causes of symptoms that can overlap with VIMS.

11. VIMS should be distinguished from visually induced vertigo as defined in the ICVD [30]. Although they can occur simultaneously, VIMS is primarily characterized by a visceral reaction (e.g., nausea, stomach awareness) whereas visually induced vertigo relates to the perception of self-motion or vection. Visually induced dizziness (VID) is recognized as a sense of spatial disorientation caused by moving or visually complex stimuli but there are no formal criteria established for VID [30]. VIMS should be diagnosed according to the criteria presented; when dizziness is the only symptom induced by visual stimulation, the designation should remain as VID. It is remains to be determined, however, whether VID and VIMS are biologically distinct disorders.

3.2.1 Criteria for Motion Sickness Disorder (MSD)
A syndrome in which Criteria A through E are met:
A. Recurrent episodes of motion sickness triggered by the same class of motion stimuli\(^1\)
   a. Probable MSD: two to four episodes
   b. Definite MSD: five or more episodes
B. Symptoms are reliably triggered by the same class of motion stimuli\(^2\)
C. Symptom severity does not significantly decrease after repeated exposure to the same class of motion stimuli\(^3\)
D. Symptoms lead to one or more of the following behavioral responses:
   a. Activity modification to abort sickness symptoms
   b. Avoidance of motion stimuli that trigger sickness
c. Negative anticipatory emotions prior to exposure to motion stimuli
E. Not better accounted for by another disease or disorder

NOTES:
1. Susceptibility to motion sickness from one type of motion may not translate to other types of motion. Therefore, motion sickness to each motion stimulus type, e.g. airplanes, automobiles, boats, should be considered separately (See Note 1, Section 3.1.1)

2. Though vehicle types vary within each class, the induction of sickness should be reasonably predictable for each stimulus class, e.g. automobiles.

3. Susceptibility to motion sickness normally habituates with repeated exposures. The repeated triggering of sickness to the same stimulus signifies an inability to habituate and is a core feature of MSD.

4. The usual behavioral response to motion sickness is to avoid sickness-inducing motion stimuli or to shorten the duration of exposure, when possible. When neither is possible, negative anticipatory emotions prior to exposure to motion stimuli may count toward a behavioral response to motion sickness.

5. An individual may be diagnosed with MSD, Visually Induced Motion Sickness Disorder (VIMSD, see Section 3.2.2), or both according to the stimulus class.

3.2.2 Criteria for Visually Induced Motion Sickness Disorder (VIMSD)
A syndrome in which Criteria A through E are met:
A. Recurrent episodes of motion sickness triggered by the same class of visual motion stimuli
   a. Probable VIMSD: two to four episodes
   b. Definite VIMSD: five or more episodes
B. Symptoms are reliably triggered by the same class of visual motion stimuli
C. Symptom severity does not significantly decrease after repeated exposure to the same class of visual motion stimuli
D. Symptoms lead to one or more of the following behavioral responses:
   a. Activity modification to abort sickness symptoms
   b. Avoidance of visual motion stimuli that trigger sickness
   c. Negative anticipatory emotions prior to exposure to visual motion stimuli
E. Not better accounted for by another disease or disorder

NOTES:
1. Susceptibility to VIMS from one type of visual motion may not translate to other types of visual motion. Therefore, VIMS to each visual motion stimulus type (See Note 1, Section 3.1.2) should be considered separately.

2. Though stimulation types vary within each class, the induction of VIMS should be reasonably predictable for each stimulus class, e.g. virtual reality goggles.
3. Susceptibility to VIMS normally habituates with repeated exposures. The repeated triggering of sickness to the same stimulus signifies an inability to habituate and is a core feature of VIMSD.

4. The usual behavioral response to VIMS is to avoid sickness-inducing motion stimuli or to shorten the duration of exposure, when possible. When neither is possible, negative anticipatory emotions prior to being exposed to visual motion may count toward a behavioral response to VIMS.

5. An individual may be diagnosed with VIMSD, MSD, or both according to the stimulus class.

3.3 Motion Sickness Susceptibility and Severity

The majority of existing motion sickness scales present composite scores made up of 1-5 categories of symptoms with subscores for each category, when applicable. Higher scores represent greater severity or susceptibility. The common elements of each scale are presented here to show the overlap in queried symptoms (per Criteria 3.1 A) (Table 1 for Severity [10][22][24][25][31]-[35] and Table 2 for Susceptibility [4][23][36]-[38].

The subcommittee recommends that each scale be used for its specific advantages to the research question or clinical application, as needed, e.g. Motion Sickness Susceptibility Questionnaire (MSSQ-Short form if several language translations are needed [23][38]; Simulator Susceptibility Questionnaire (SSQ) if VIMS is the focus [24]. For brief assessments in clinical settings, patients may be asked how frequently they feel sick during motion on a simple Likert scale, e.g. “Never,” “Sometimes,” “Often,” or “Always.” Subjective susceptibility assessments on simple scales have strong correlation with more detailed assessments that query sickness to specific triggers [23][38][39]. Similarly, activity avoidance as a measure of susceptibility can be ascertained by querying how usual it is for the patient to avoid certain motion stimuli in order to avoid becoming motion sick, e.g. “Never,” “Sometimes,” “Often,” or “Always.”

While motion sickness can be induced in nearly all individuals with a sufficiently strong stimulus, there are individuals at the extremes of the population distribution of susceptibility. These may occur in the setting of normal or abnormal central or peripheral vestibular function. The distribution curve of susceptibility is linear up to about the 75th percentile of susceptibility and flattens out on the high end of scores, e.g. on the MSSQ-Short form, out of a total possible score of 54, a score of 11=50th percentile, 19=75th percentile, 27=90th percentile, 31=95th percentile, indicating that only a small proportion of individuals are severely susceptible to motion sickness, i.e. 90% of individuals score 0-27 while 10% of individuals score 28-54 on the MSSQ scale [37].

Norms may be different depending on the age and sex distribution of the population sampled, which should be considered when working with non-representative populations [39][40]. Motion sickness susceptibility changes with age with a large transition occurring during adolescence. Therefore, defining whether the symptoms refer to the ≤12 year range or the >12 year range will help in accurate communication of current status and aid in determination of prognostic variables. Based on a simplified scoring system of the MSSQ [23][38] adopted from Reason and Brand, 1975 [4], the Pearson correlation between childhood (Part A) and adult motion sickness scores (Part B) is $r = .65$ [23][38].

Despite the similarity in etiology and symptomatology, susceptibility to motion sickness is not a reliable predictor of susceptibility to VIMS. For instance, older adults are more susceptible to VIMS but not to
motion sickness than younger adults [39]. Nausea and vomiting are typically less common in VIMS, whereas ocular motor issues (e.g., eyestrain, blurred vision) and headache are more common in VIMS than in motion sickness caused by self-motion [23][38][41].

Motion sickness severity and susceptibility can be modulated by reducing exposure to sickness-inducing stimuli or, conversely, by engaging in measured amounts of motion exposure to induce habituation. Habituation may be stimulus specific, however and may not generalize across motion types or situations. Environmental adaptations such as roll-stabilizers on ships, active suspension in cars, or virtual reality displays with short head motion response lags can reduce the chances of inducing motion sickness. Interventions such as habituation exercises (eye-head motion, repeated exposure [17]), pharmacologic pretreatment (anti-muscarinic, anti-histaminic, anti-cholinergic medications [42]); non-pharmacological treatments (music, smells [43]), and behavioral techniques (breathing exercises, meditation [44]) can all adjust severity.

3.4 MSD and VIMSD impact
Motion sickness that is mild, easily avoidable, or has no functional impact is common among individuals with normal vestibular function and should not be considered a disorder. However, motion sickness susceptibility that affects activities of daily living such as meeting basic transportation needs or fulfilling obligations to family, employment, or social functions can have physical and mental health consequences. Even in otherwise healthy individuals, motion sickness can reduce work or school productivity through direct effects from being ill, avoidance of sickness-inducing activities, or sedation induced by pharmacological motion sickness remedies [10][45][46]. Nausea, lethargy, drowsiness, and increased interoceptive awareness can shift attention away from critical tasks while vomiting can lead to dehydration or aspiration [27][47].

Formal disability scales may be paired with existing susceptibility scales to quantify disability due to MSD or VIMSD. As an example, the Sheehan Disability Scale quantifies impact on work, family, or social function on a 10-point scale along with a query of loss of work time or productivity due to symptoms [48]. Alternatively, a basic impact assessment that queries whether work, social, family, or travel difficulties are experienced (Yes or No) due to symptoms can be used as a quick assessment, e.g, the Social Work Impact of Dizziness short form adapted for motion sickness [49][50].

Even if motion sickness does not create a disability, it should be recognized as a clinical entity that deserves medical attention. In most cases, education, exposure minimization, short-term use of anti-motion sickness medications, or simple habituation exercises may be employed to avoid motion sickness contributing to morbidity and evolving into a disorder.

4. Motion Sickness Clinical Features
4.1 Prevalence: The prevalence of motion sickness in childhood has been estimated at 35-43% prior to puberty and 25% in young adults; it is a frequent problem in 14% of adults younger than 30 years old and 7% of adults 61 years old or older [51]-[53]. The prevalence of VIMS ranges widely depending on the stimulus type and the visual content, with rates varying from 1% [54] to 60% [55] to 80-95% [56][57].

4.2 Demographics: Susceptibility to motion sickness changes with age. Infants are fairly resistant to motion sickness until about age two, at which point motion sickness susceptibility rises and peaks
between the ages of 7-12 years and declines thereafter and throughout adulthood [15][51][58]. Susceptibility gradually declines with age but may increase in a small proportion of individuals [10]. Prevalence estimates should consider that people might self-restrict their behavior and avoid situations that provoke motion sickness if they are aware of their elevated susceptibility.

In contrast, an increase in susceptibility to VIMS as a factor of age has been well-documented, with older adults often reporting more VIMS than younger adults [39][59][60]. Sex differences in VIMS have been observed in some studies [5][60]-[63], but it remains unclear whether women actually experience more VIMS or are more open about reporting it than men [62]. A variety of other factors, including technical manipulations such as the size of the field-of-view or individual habits such as video game experience can affect the occurrence of VIMS [65][66].

Motion sickness prevalence and severity are higher in females than males by as much as a factor of two, but estimates vary among studies depending on stimulus type, endpoint studied, age, ethnicity, and hormonal status [23][53][67]-[71]. Specifically, even when the incidence of motion sickness is reported to be similar between women and men, episodes of vomiting and duration of sickness days are higher in women [71]. As a perspective, however, the effect of sex is about one-third that of age [72][73].

4.3 Genetics: Heritability for motion sickness in females show a 0.69 concordance in monozygotic twins and 0.44 concordance in dizygotic twins for childhood motion sickness yielding a heritability estimate of 70% [51]. A large genome-wide association study involving 80,494 individuals with carsickness found 35 single nucleotide variants at genome-wide significant levels. The top ten genes involved in these regions included: PVRL3, GPD2, ACO1, AUTS2, GPR26, UBE2E2, CBLN4, MUTED, LINGO2 and CPNE4 [53]. A genetic risk score using the number of risk alleles found on each individual for these 35 variants could be used to anticipate motion sickness susceptibility. These genes involve a wide variety of functions such as brain, eye, and ear development and even insulin resistance. Some of these loci overlap with genes in individuals who experience dizziness, post-operative nausea and vomiting, altitude sickness, morning sickness, indigestion to dairy, and headache after red wine [53].

Studies that have assessed motion sickness susceptibility by questionnaire, rotation of the body with head pitching, and exposure to optokinetic drums have reported heightened susceptibility in individuals of Chinese compared to European descent with the majority of the differences occurring between women in the two groups [66][74]. These differences persist in American-born children of the Chinese parents, supporting at least a partial genetic component to motion sickness susceptibility [74].

4.4. Other Modifying Factors: Hormonal factors such as the use of oral contraceptives, menstruation, pregnancy, and cortisol levels correlate with motion sickness susceptibility in women [72][75][76]. Other modifying factors may include baseline autonomic tone, glucose levels, and physical fitness [77]-[79].

4.5 Associated Syndromes

4.5.1 Migraine: Symptoms of migraine and motion sickness overlap in many domains, e.g., nausea, stomach awareness, and headache (For review see [69] and [80]). Motion sickness is self-reported in at least 50% of migraine headache sufferers with motion sickness in childhood correlating with eventual development of migraine headaches in adolescence and adulthood [23][58][69][81]. These associations suggest that neural pathways for nausea and emesis are particularly sensitive in individuals
with migraine [23]. Scalp tenderness and nausea during optokinetic stimulation increase more for those who experience migraine than for those who do not [82]. Motion sickness induced by OVAR is especially intense in individuals with migraine [26]. This type of motion sickness may be blunted by rizatriptan, a migraine abortive medication [83][84]. In contrast, VIMS does not appear to be mitigated by pre-treatment with rizatriptan [85]. Individuals with migraine headaches experience both motion sickness and VIMS to a higher degree than those without migraine, but the correlation between motion sickness and VIMS in this group is weak [86].

4.5.2 Structural vestibular disorders: Loss of peripheral vestibular function significantly raises the threshold for motion sickness and VIMS [26][87]-[89]. Congruently, patients with chronic vestibular loss (unilateral and bilateral) report less motion sickness on clinical motion sickness ratings than healthy controls. In comparison, patients with BPPV show a non-significant difference in motion sickness susceptibility [58]. Patients with vestibular neuritis can experience either an increase or a decrease in motion sickness susceptibility after the onset of their disorder, a difference attributable to whether the loss has been compensated [26][41]. Two studies have shown that individuals with Meniere disease are more susceptible to motion sickness than healthy controls but are not as susceptible as those with migraine or vestibular migraine [90][91]. There is no correlation between the degree of caloric asymmetry in vestibular patients and scores on a clinical motion sickness questionnaire. In contrast, patients with vestibular disorders without vestibular loss report higher degrees of motion sickness than controls [29][58]. About 10-minutes of exposure has been shown to distinguish between individuals of different susceptibility to motion sickness in laboratory settings (vestibular neuritis, bilateral vestibulopathy, vestibular migraine) [26].

5. Motion Sickness Laboratory Examinations
Motion sickness may be induced in laboratory settings in order to probe the components of susceptibility, but these tests are not used for clinical diagnostic purposes. Laboratory studies may elucidate why there may be differences in motion sickness sensitivity to different stimuli, however. Correlations between the MSSQ with laboratory-induced nausea by cross-coupled stimulation has been reported to be between 0.14 and 0.58 with generally higher correlations being seen for vertical translational oscillations than horizontal oscillations [23]. Of the translational planes, motion through the body-referenced X- or Y-axes (fore-aft or side-to-side motion when upright, respectively) are more provocative than the body-referenced Z-axis, (up-down) but motion direction with respect to the gravity vector (e.g. vertical versus horizontal) is a less important factor [92].

An area of controversy is whether a smaller phase lead of the vestibulo-ocular reflex (VOR), which indicates enhanced velocity storage, is related pathophysiologically to motion sickness susceptibility as found in some laboratories ([92]-[96]) but not replicated in others ([73][83]). It has thus been proposed that the absolute value of the VOR may not be the relevant marker of motion sickness susceptibility but rather the ability to modify the VOR in response to varying motion sickness-inducing stimuli that is key to whether motion sickness develops to any given stimuli [10][73]. The relationship between motion sickness susceptibility and adaptability of vestibular responses has also been supported by lower cervical vestibular evoked myogenic potential (VEMPS) thresholds correlating with the ability to habituate to seasickness; this may be attributable to the wider potential range of adaptive responses to motion stimuli with lower VEMP thresholds [97].

6. Differential Diagnosis
Because motion sickness susceptibility declines with age, an adult patient presenting with increasing susceptibility to motion should be evaluated for underlying causes of a lowered threshold for nausea. This evaluation may include an assessment for migraine, endocrine abnormalities, ocular misalignment, and other central nervous system disorders. Head motion-induced discomfort should be suspected as being secondary to an uncompensated vestibular asymmetry. These cases should be evaluated with vestibular laboratory testing.

7. Future Directions
The presentation of these criteria for motion sickness acknowledges that there are still critical information gaps that need to be filled in order to determine a clear line between motion sickness as a normal human response to movement and motion sickness as a disorder. The difficulty in determining this line in no small part lies in the large number of variables that contribute to susceptibility and morbidity, one of the most important being activity modification.

A challenge in motion sickness research has been the rapid technological advancements in transportation and entertainment options that have inadvertently created an environment of increasing motion stimuli in both novelty and number [98][99]. It remains to be determined whether human beings may be able to push current boundaries of motion tolerance through enhanced habituation exercises, manipulation of vestibular input, cognitive enhancement techniques, pharmacological therapies, or noninvasive brain stimulation methods. Prediction of susceptibility based on demographics, hormonal rhythms, genetics, and physiological status may lead to optimization of environments that reduce stimulus intensity and morbidity.

Toward those ends, criteria for motion sickness have been proposed here to promote clearer communication among clinicians and investigators. It is expected that criteria for motion sickness will evolve as data on epidemiology, natural history, development of biomarkers, and assessments of future health consequences are methodically acquired.

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References


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<td>SSQ</td>
<td>SR</td>
<td>NP</td>
<td>MSAQ</td>
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<td>Visual Simulator</td>
<td>Single Item</td>
<td>Composite w/9 parts Self</td>
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Table 1: Motion Sickness Severity Scales

*NOTE: This 6-point scale was used as a convention in the Royal Air Force for some time prior to appearance in publications.
### Table 2: Motion Sickness Susceptibility Scales

<table>
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<tr>
<th>Known as</th>
<th>Motion Sickness Questionnaire [4][36]</th>
<th>Pensacola Motion History Questionnaire [37]</th>
<th>Motion Sickness Susceptibility Questionnaire [23]</th>
<th>Motion Sickness Susceptibility Questionnaire [38]</th>
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<tbody>
<tr>
<td>Shorthand</td>
<td>MSQ</td>
<td>MHQ</td>
<td>MSSQ-Long (54-items)</td>
<td>MSSQ-Short (18-items)</td>
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<tr>
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<td>Composite w/2 parts</td>
<td>Composite w/2 parts</td>
<td>Composite w/2 parts</td>
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<td>Adult (since 12 yrs or last 10 yrs)</td>
<td>Part B Adult (last 10 yrs)</td>
<td>Part B Adult (last 10 yrs)</td>
<td>Part B Adult (last 10 yrs)</td>
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<td>Queries amount of exposure, episodes of feeling sick and nauseated, and episodes of vomiting in each of 9 triggers</td>
<td>Queries amount of exposure, episodes of feeling sick and nauseated, and episodes of vomiting in each of 9 triggers</td>
<td>Queries episodes of feeling sick or nauseated during each of 9 triggers</td>
<td>Queries episodes of feeling sick or nauseated during each of 9 triggers</td>
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</table>